Case Report

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# Cocaine-induced Stroke in a First-time User

Internal Medicine Section

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## ABSTRACT

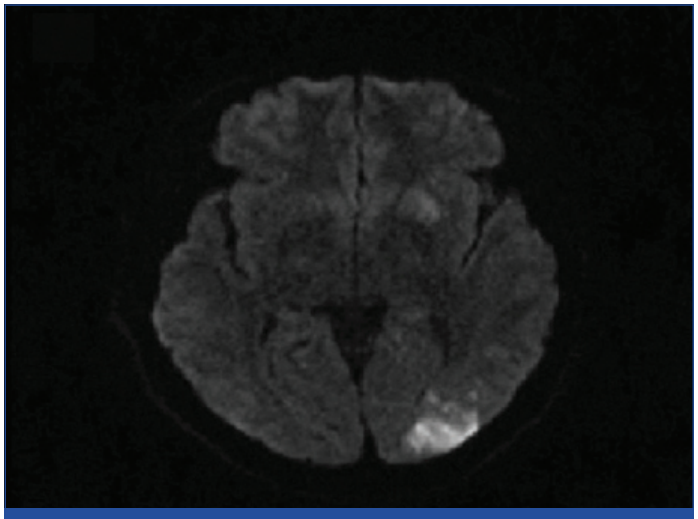
Cocaine is a widely used recreational drug that functions by inhibiting the reuptake of neurotransmitters in the brain, contributing to feelings of euphoria. Its use can result in stroke primarily through the mechanism of vasospasm, especially in the acute phase. Here, we present a case of a 47-year-old previously healthy male with no family or personal risk factors for stroke, who presented with acute neurological deficits after his first intranasal cocaine use with brain imaging showing scattered areas of restricted diffusion in his left Middle Cerebral Artery (MCA) territory and MCA/Posterior Cerebral Artery (PCA) watershed zone consistent with vasospasm. This case highlights the importance of considering a toxicology screen for cocaine even in the absence of history when working up a young patient presenting with stroke-like symptoms.

**Keywords:** Cocaine, First time cocaine use, Headache, Stroke

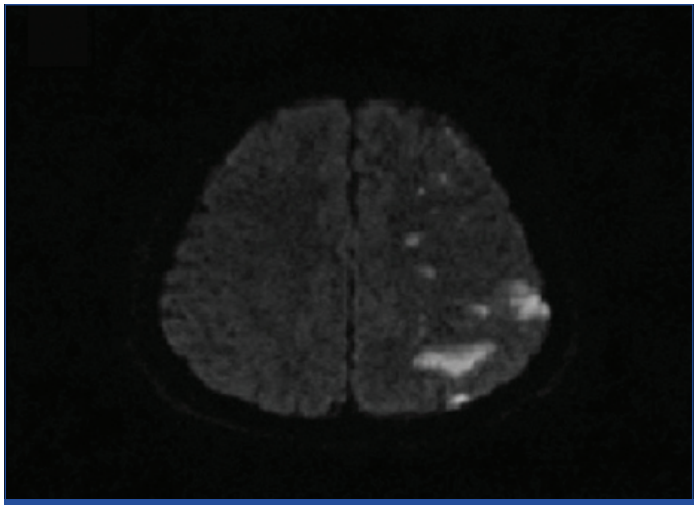
## CASE REPORT

A 47-year-old previously healthy hispanic male, with no family history of stroke, presented to the ED a few hours after his first intranasal cocaine use with acute onset headache, bilateral blurry vision, lethargy, confusion, and difficulty speaking, as well as a low-grade fever. His neurological exam on admission was remarkable only for expressive aphasia and mild dysarthria bilaterally. Routine laboratory profiles, including a lipid profile and HbA1c, were normal. A chest X-ray and echocardiogram were unremarkable. Blood cultures showed no growth to date after five days and the toxicology report was positive only for cocaine.

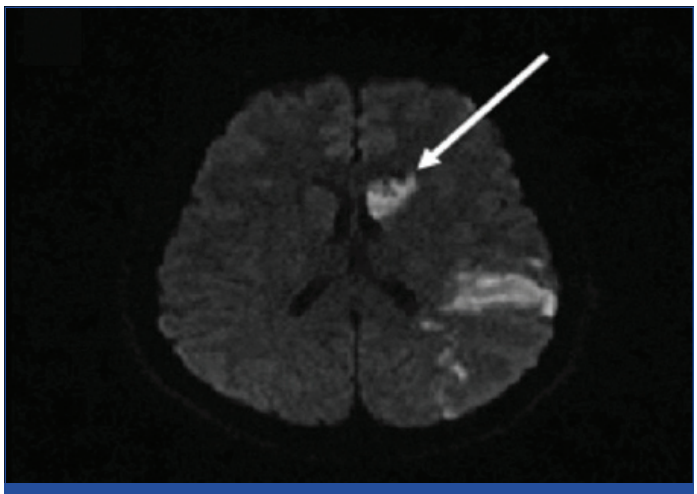
Computed tomography without contrast and a brain MRA without contrast were unremarkable. A brain MRI showed areas of restricted diffusion in the left occipital lobe in the MCA/PCA watershed zone [Table/Fig-1] and scattered foci of restricted diffusion in the left frontal, parietal, and temporal lobes within the MCA territory [Table/Fig-2,3], as well as the head of the left caudate (arrow in [Table/Fig-3]. Of note, the patient's neurological exam evolved on day 3 of his hospital stay to include 3/5 strength in his right hand with the remainder of the neurological exam unchanged. On hospital day 4 (the day of discharge), his right hand strength improved to 4/5 on neurological exam and he was able to state his name and answer questions in short phrases, a notable improvement in his expressive aphasia since admission. He was discharged to a rehabilitation facility, but was subsequently lost to follow-up. Given that his workup identified only cocaine use as a risk factor for stroke; his findings are most consistent with severe vasoconstriction secondary to cocaine use, which likely explains the neurological gains observed on admission without specific intervention.



**[Table/Fig-1]:** A brain MRI without contrast showing areas of restricted diffusion in the left occipital lobe in the MCA/PCA watershed zone.



**[Table/Fig-2]:** A brain MRI without contrast showing scattered foci of restricted diffusion in the left frontal, parietal, and temporal lobes within MCA territory.



**[Table/Fig-3]:** The arrow highlights an area of restricted diffusion in the head of the left caudate in a brain MRI without contrast.

## DISCUSSION

While much of our healthcare system's recent focus has been on the rising use of opioids, cocaine is one of the most commonly abused recreational drugs, second only to marijuana [1]. It blocks the reuptake of norepinephrine and dopamine, which contributes to feelings of euphoria and its excitatory effects [1].

The mechanism by which cocaine can cause stroke is believed to be multifactorial and includes vasospasm, cardioembolism, and altered cerebral auto-regulation secondary to hypertensive surges [2]. Evidence from inpatient patient samples suggests that vasospasm is particularly linked with cocaine use [3].

Cocaine-related strokes, both ischaemic and haemorrhagic, have been well-described in the literature. Ischaemic strokes have been described in the bilateral hippocampi in a 44-year-old male [4], in the bilateral posterior inferior cerebellar arteries and hippocampus in a middle-aged female [1], and in the globus pallidus bilaterally (with subsequent hemohaemorrhagic transformation) in a 30-year-old female [5]. Even despite normal blood pressure, there has been a case report of intracerebral hemohaemorrhage in a middle-aged cocaine user [6]. To our knowledge, ours is the first case report demonstrating areas of restricted diffusion within watershed zones and the caudate nucleus.

Current literature suggests a critical temporal relationship between cocaine use and stroke risk in young patients. A population-based case-control study design identified that acute cocaine use in the previous 24 hours was strongly associated with increased risk of stroke (age-sex-race adjusted odds ratio, 6.4; 95% confidence interval, 2.2-18.6) in patients aged 15-49 years [7].

Cocaine use is associated with higher in-hospital mortality and more frequent rehabilitation requirements compared to non-cocaine-induced stroke [3]. In addition to medical management, behavioural intervention is critical in the treatment of cocaine-induced stroke as a method of prevention in order to discourage the patient from the use of cocaine in the future.

## CONCLUSION

Cocaine use is common among young patients given its euphoric effects. The mechanism by which is causes stroke is largely attributed to vasoconstriction, resulting in potentially broad neurological territories of involvement. The differential of cocaine-induced stroke should be kept in mind when patients present with ischaemic changes that do not confine to a vascular territory, especially in younger patients. A toxicology screen is therefore an important addition to the stroke work-up in a young patient.

## REFERENCES

[1] Mullaguri N, Battineni A, Narayan A, Guddeti R. Cocaine induced bilateral posterior inferior cerebellar artery and hippocampal infarction. Cureus. 2018;10(5):e 2576.
[2] Treadwell SD, Robinson TG. Cocaine use and stroke. Postgraduate Medical Journal. 2007;83(980):389-94.
[3] Desai R, Patel U, Rupareliya C, Singh S, Shah M, Patel R, et al. Impact of cocaine use on acute ischemic stroke patients: insights from nationwide inpatient sample in the United States. Cureus. 2017;9(8):e1536.
[4] Connelly KL, Chen X, Kwan PF. Bilateral hippocampal stroke secondary to acute cocaine intoxication. Oxford Medical Case Reports. 2015;2015(3):215-17.
[5] Kadi R, Rumy A, Stadnik T, Cannie M, Mabiglia C, Divano L. Bilateral lesions of the globus pallidus in a young woman. Journal of the Belgian Society of Radiology. 2014;97(2):118-20.
[6] Siniscalchi A, Lentidoro W, Pisanil E, Sarro GD, Gallelli L. Intracerebral hemorrhage in a middle-aged cocaine user despite normal blood pressures. The American Journal of Emergency Medicine. 2017;35(3):516.e3-516.e4.
[7] Cheng YC, Ryan KA, Qadwai SA, Shah J, Sparks MJ, Wozniak MA, et al. Cocaine use and risk of ischemic stroke in young adults. Stroke. 2016;47(4):918-22.

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